Investigation of the Relationship between Pressure and Injury by Impacting *Ex Vivo* Perfused Spleens

Undergraduate Research Thesis

Presented in Partial Fulfillment of the Requirements for Graduation with Honors Research Distinction

By

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Abstract

Motor vehicle crashes (MVCs) can be fatal and may induce permanent disability to the individuals involved. MVCs may result in injury to different body regions, but in particular, the abdomen is a site of life-threatening injuries due to housing vital body organs. The spleen is the second most injured abdominal organ following the liver, but the major causation of the splenic injuries remains largely unknown. Since the spleen is believed to have viscoelastic characteristics, sudden change in pressure during MVCs might be a source of these injuries. The main objective of the present study is to investigate the relationship between impact-induced pressure change and corresponding injuries in an *ex vivo* organ experimental model. Historically, there have been limited attempts to impact a pressurized *ex vivo* spleen; therefore, this study is one of the first times impact based tests are used to relate pressure changes to splenic injuries.

The *ex vivo* spleens were instrumented with two miniature pressure sensors inside of foley catheters located in the splenic artery and vein. A perfusion system was developed to reproduce the physiological pressure of *ex vivo* spleens between 60-120 mmHg at steady state. Using an electromagnetic or hand trigger mechanism, an impact plate (23.4 kg) was dropped from varying heights to vary impact energies. The *ex vivo* spleens were impacted up to three times until gross injuries to the spleen occurred. The injuries were assessed according to the Abbreviated Injury Scale (AIS), and the probability of injury was plotted against pressure, velocity, and the rate of pressure change in the artery,

$\dot{P}_{artery}$. Gross injuries with AIS ≥3 to the *ex vivo* spleens occurred from pressures around 11 psi and from $\dot{P}_{artery}$ after 1057 psi/sec. The findings suggest that pressure and $\dot{P}_{artery}$ correspond well with injury severity, whereas velocity does not correspond well with injury severity. This study serves to show that pressure and $\dot{P}_{artery}$ may be used to predict injuries to the spleen during impact scenarios. Such relationships will help to better define abdominal injury criteria when simulating impacts to the abdomen of dummies or human finite element models.

Dedication

Dedicated to my family, Han Young Ryu, Sang Hee Lee, and Hyunsu Christina Ryu.

A special dedication to the individuals who have donated their bodies for this project.

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This project would have been impossible without the help of my graduate student mentor, Rakshit Ramachandra. His knowledge, encouragements, and guidance have motivated me to focus on and finish my project.

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Ryu Y, Ramachandra R, Bolte JH. "Investigation of the Relationship between Pressure and Injury by Impacting *Ex Vivo* perfused Spleens" presented at OSU Spring Undergraduate Research Forum (March 21, 2014)

Bolte JH, Bing JA. "Analysis of comfort in forward-facing vs. rear-facing child restraint systems" Presentation at Center for Child Injury Prevention Studies, Ryu Y's contribution was acknowledged (April 19, 2014 and November 20, 2014)

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FIELDS OF STUDY

Major Field: Biomedical Engineering

Table of Contents

List of Tables

List of Figures

Chapter 1: Introduction

Motor vehicle crashes can be fatal and may induce permanent disability to the individuals involved. Automotive crashes may result in injury to different body regions, but in particular, the abdomen is a site of life-threatening injuries due to housing vital body organs [1]. Organs such as the liver, spleen, and kidney are most often injured abdominal organs in motor-vehicle collisions [2]. The spleen is the second most injured organ following the liver; the percentage of occupants with splenic injuries is estimated to be 23.1% following 39.2% estimated percentage of liver injuries [3]. Additionally, seatbelt use in nearside crashes has proven to be unhelpful for spleens [4]. These statistics indicate that blunt impacts to the abdominal region in motor-vehicle crashes could be the main cause of liver and spleen injuries. However, less research has been performed on the spleen than the liver. Since the spleen tissue has viscoelastic characteristics, sudden change in hydrostatic pressure during motor vehicle collisions might be a source of these injuries. There is a need to define the major causation of the splenic injuries in automotive crashes. The authors hypothesize that the pressure is the major source of splenic injury in motor vehicle collisions. The outcome of this research may help develop mechanisms to prevent major injury to the spleen and the organs around it.

The present study will investigate the correlation between damages in the spleen and pressure changes in the spleen due to blunt impacts. Historically, there have been little attempts to pressurize an *ex vivo* spleen by impacting it; therefore, this experiment

will be one of the first times impact based tests are used to pressurize the spleen to relate pressure and damage to the spleen. A technique to apply blunt impacts to *ex vivo* spleens was selected, and a series of experiments was conducted to evaluate whether pressure change is significantly associated with spleen injury severity defined by the Abbreviated Injury Scale (AIS).

The main objective of the present study is to investigate the relationship between impact-induced pressure change in the spleens and corresponding damage to the spleen in an *ex vivo* organ experimental model.

1.1 Overview of the Spleen Anatomy

The spleen is an organ located left of the stomach in abdominal viscera. It is under the diaphragm, surrounded by other abdominal organs, and positioned near 9^{th} to 11^{th} ribs [6]. The primary function of the spleen is the filtration of blood to identify and fight antigens that may be present in the blood [5, 6]. The spleen receives its blood from the splenic artery (A), a branch of the celiac trunk, and drains its blood to the splenic vein (V), which eventually drains to the hepatic portal vein (Figure 1). It consists of two materials, the red and white pulp. The red pulp is considered to be the majority of the splenic tissue, and the white pulp is looked to be white due to the white blood cells for the immune system [6]. Because the spleen is highly vascular, the organ receives more than 5% of the cardiac output and is reddish purple in color [7].

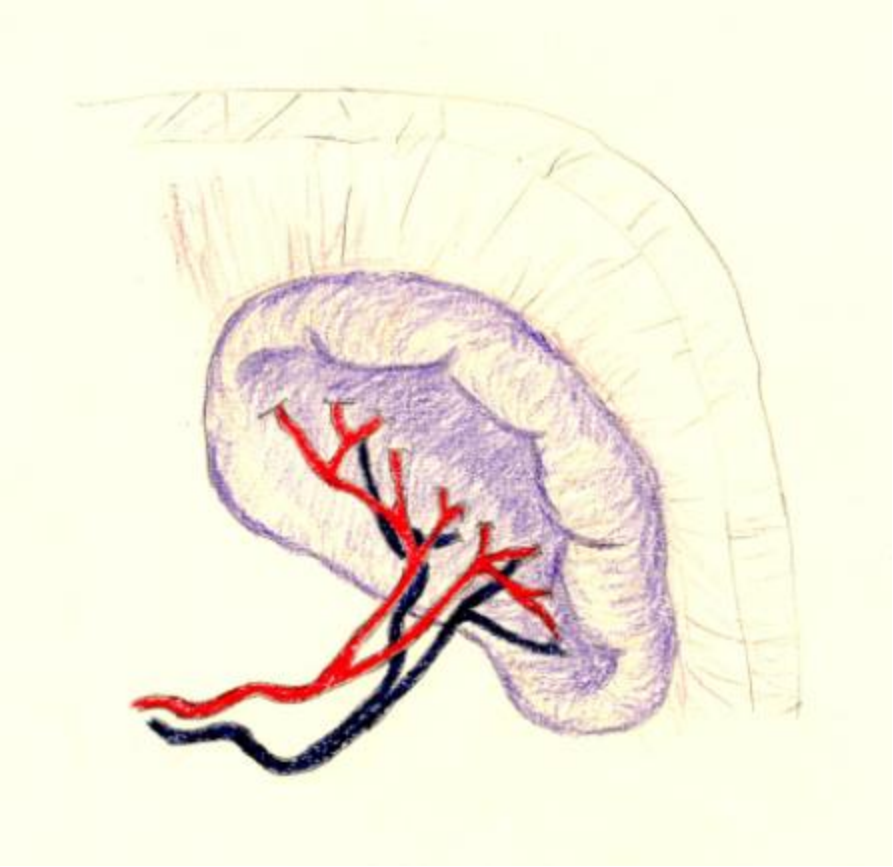


Figure 1. Spleen anatomy. The red and blue vasculatures represent the splenic artery and the splenic vein, respectively.

Chapter 2: Methods

2.1 Instrumentation

Autopsies were performed on post-mortem human subjects (PMHS) to obtain spleens for testing, and the *ex vivo* spleens were inspected for any visible damages prior to testing. The height, length, and depth of the spleens were measured (Table 1). The splenic A and splenic V were identified. The *ex vivo* spleens were then equipped with foley catheters (14-16 FR) and a miniature pressure-measuring sensor (Model SPR-524, Millar Instruments, Houston, TX) with a diameter of 1.2 mm, a natural frequency over 10 KHz, and a range of 386 kPa in the splenic A and V each (Figure 2) [8,9]. Since the sensors were designed for constant temperature, non-flow, in-fluid applications, the sensors were pre-soaked to minimize inaccurate readings of pressure measurements [8,9].

Table 1. Subject Characteristics

Subject	Height (cm)	Width (cm)	Length (cm)	Weight (g)
Spleen01	1.8	7.5	12.8	141
Spleen02	4	6.5	8.5	---
Spleen03	2	8.8	9	104
Spleen04	3.5	8	14.5	242

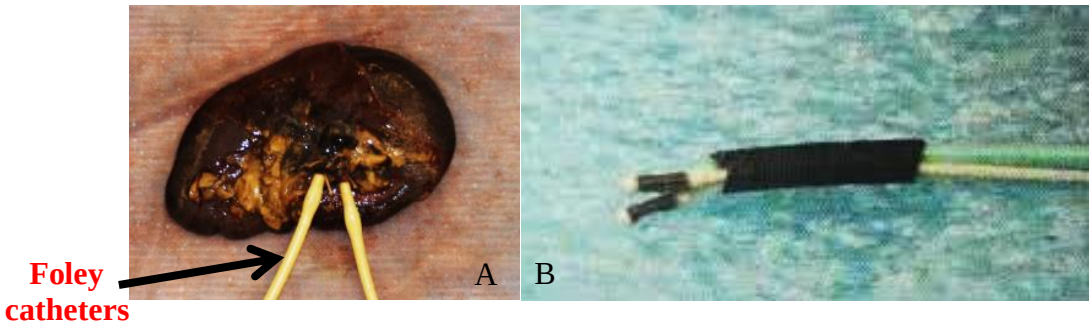


Figure 2. *Ex vivo* spleen instrumentation (a) spleen with foley catheters (b) Millar pressure sensors

2.2 Drop Tower System

Alongside the instrumentation of the *ex vivo* spleens, a drop tower system was set up to impact the spleens (Figure 3a). The system closely followed the experimental setup used by Sparks in the liver injury study [8]. Two accelerometers (Endevco, Irvine, California) and two load cells (Vishay Precision Group, Malvern, Pennsylvania) were mounted to the impact plate. The two load cells were arranged to be 120° apart from each other and the *ex vivo* spleens were placed under the drop plate with their highest point aligned with the load cell on the center of the impact plate to evenly distribute the load [8,9]. Using an electromagnetic trigger mechanism or a hand dropper, an impact plate made of steel and aluminum (23.4 kg) was dropped from varying heights of 1 m/s, 2 m/s, and 3 m/s (Table 1).

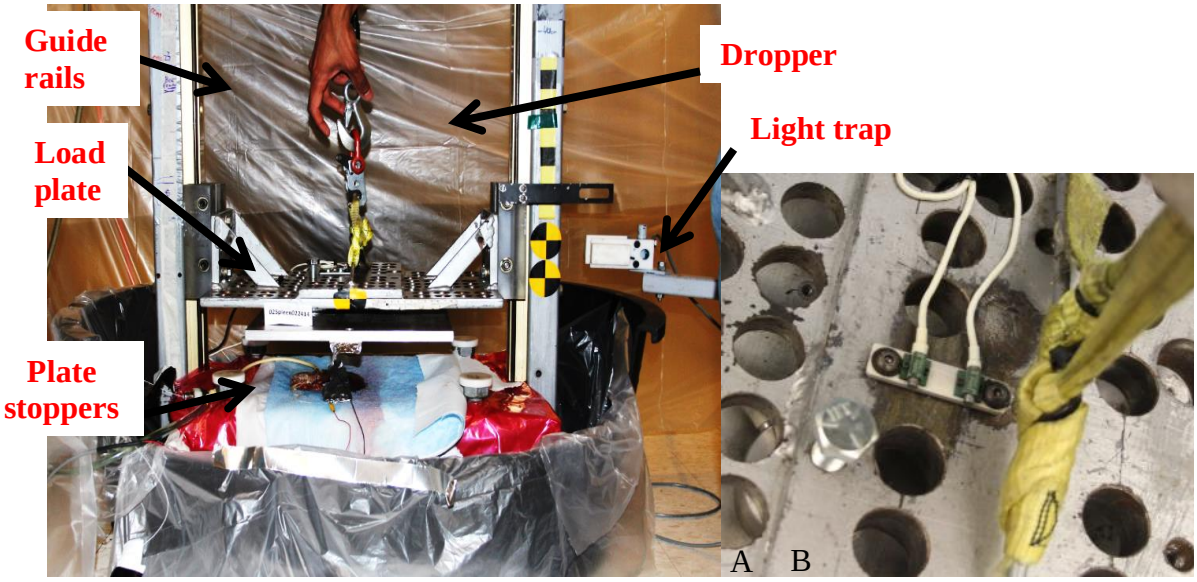


Figure 3. Drop tower system (a) drop tower setup with an ex vivo spleen. The load plate is able to be stopped at 70% of the height of the spleen due to the four plate stoppers. (b) accelerometers on the load plate

Initial impact heights were calculated, and the velocities were experimentally determined using foam tests and light trap to achieve the desired impact energies to produce accurate velocity (Table 2, Figure 4) [8]. The *ex vivo* spleens were impacted up to three times and compressed up to 30% until visible damages to the spleen occurred. In order to ensure that the data acquisition system was synchronized with the first contact of the impact, contact sensors were mounted on the bottom of the plate and the highest point of the *ex vivo* spleens.

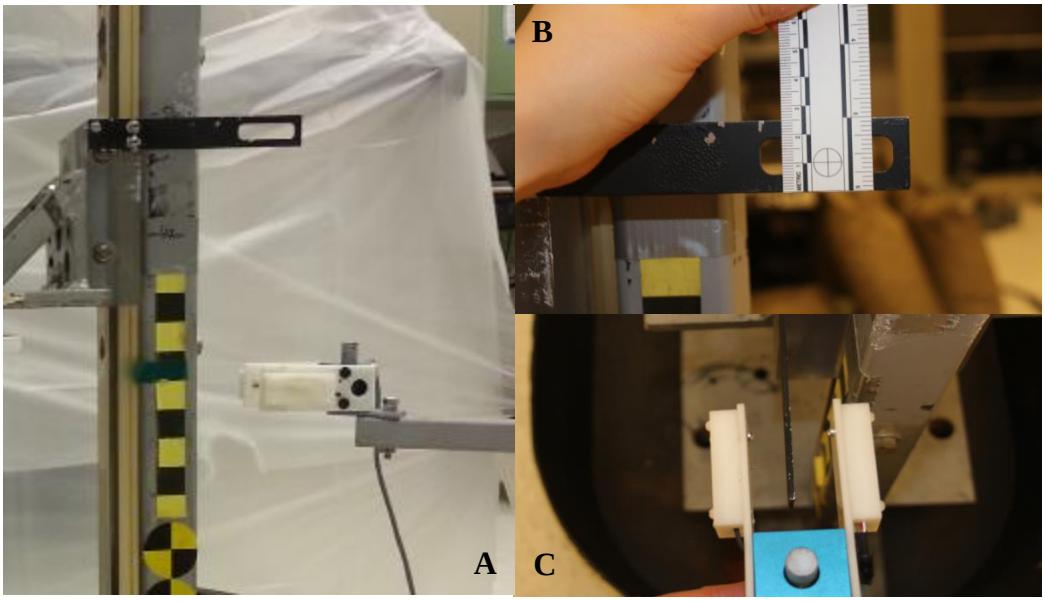


Figure 4. (a) Light trap setup. (b) A black bar attached to the load plate with a hole with a height of 2.54 cm. (c) Top view of the light trap and the black bar.

Table 2. Test matrix including the number of subjects and the maximum number of trials with drop velocities and drop heights**.**

Subject	Trial 1	Trial 2	Trial 3
N_{1-4}	Low velocity impact	Median Velocity impact	High Velocity impact
	1 m/s (height of 8 cm)	2 m/s (height of 23 cm)	3 m/s (height of 47 cm)

2.3 The Perfusion System

The perfusion system was developed to reproduce the physiological pressure of *ex vivo* spleens between 60-120 mmHg at steady state (Figure 5) [7]. Each spleen were perfused through its splenic A and V with reservoir of normal saline solution, a sterile solution of sodium chloride (NaCl) in water at room temperature until a nominal arterial pressure was maintained [8]. Each *ex vivo* spleens were perfused until visible rise in the size of the spleen was detected [8].

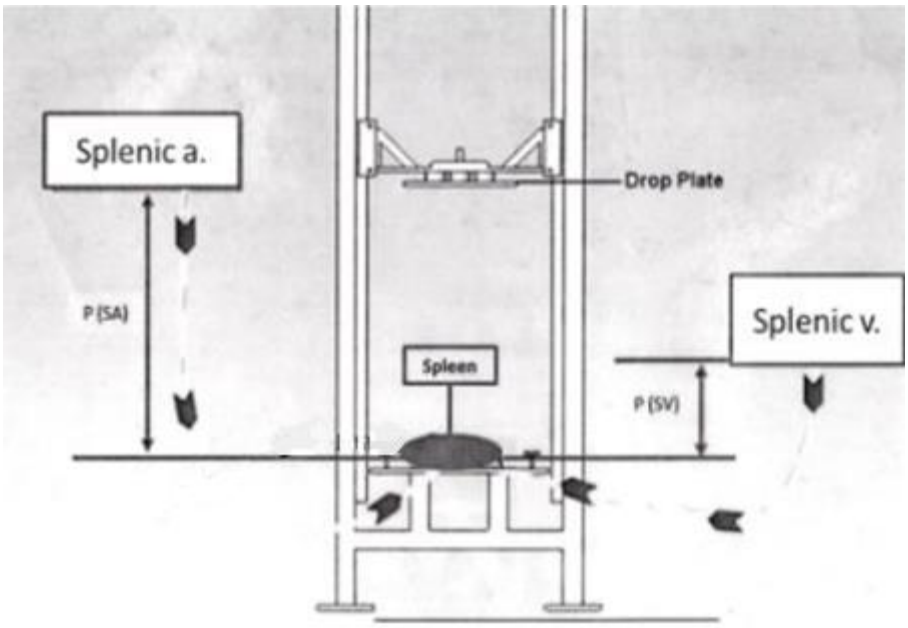


Figure 5. Schematic of the perfusion system. Buckets including Saline reservoirs were set at heights of 53.5 in H_2O and 4.81 in H_2O for splenic A and splenic V, respectively. P(SA) and P(SV) indicates pressure in splenic A and V. IVC indicates Inferior vena cava reservoir.

2.4 Impact Analysis

After completing the impact tests of *ex vivo* spleens, the damage on the surface of the spleen were assessed according to the range of AIS. The injury scale for the spleen is described in Table 2. The vascular pressure of the spleen during the impact was recorded.

Table 3. Abbreviated Injury Scale for the spleen [9]

Injury	AIS Score	Description
2	Moderate	(Laceration) Capsular tear, <1 cm parenchymal depth, 1-3 cm parenchymal depth which does not involve a trabecular vessel
3	Serious	(Laceration) > 3 cm parenchymal depth or involving trabecular vessels
4	Severe	(Laceration) Laceration involving segemental or hilar vessels producing major devascularization (> 25% of spleen)
5	Critical	(Laceration) Completely shattered spleen

2.5 Data Processing

Two different data acquisition system were used, the TDAS G5 (Diversified Technical Systems, Seal Beach, California) and the Yokogawa system (Yokogawa Electric Corporation, Sugar Land, Texas). Data will be collected at a sampling frequency of 20 kHz. The rate of arterial pressure change during the compression was calculated to be compared with injury risk (Equation 1).

$$\dot{P}_{artery} = \frac{P_{peak\ of\ compression} - P_{beginning\ of\ compression}}{Time_{peak\ of\ compression} - Time_{beginning\ of\ compression}} \quad (1)$$

Impact velocities were calculated from the light trap (Equation 2).

$$v = \frac{\text{height of the bar}}{Time_{top\ of\ bar} - Time_{bottom\ of\ bar}} = \frac{0.0254\ m}{t_{top} - t_{bottom}} \quad (2)$$

From the impact velocities and the load plate mass, impact energies were determined using Equation 3, where m = mass of the plate and v = velocity.

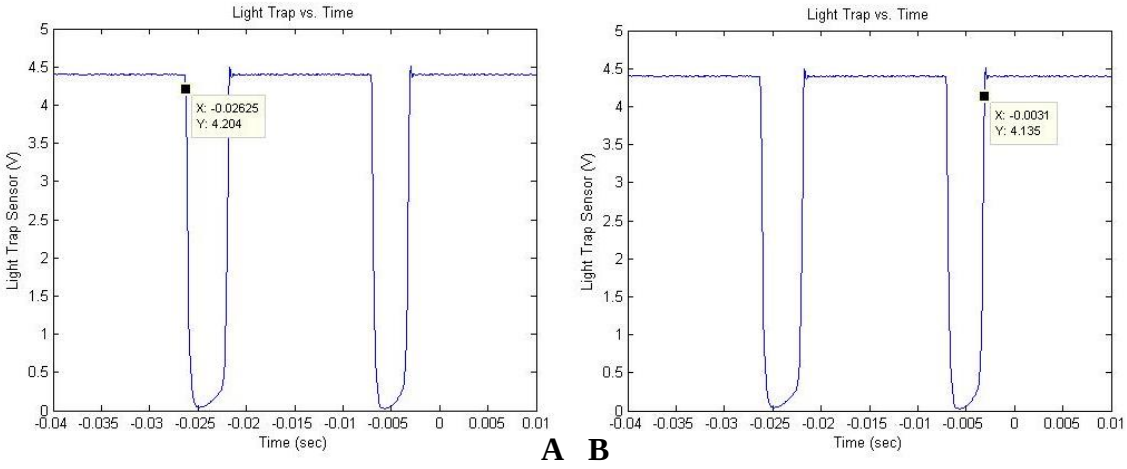
$$\text{Energy} = \frac{1}{2}mv^2 \quad (3)$$

Using two data processing software, MATLAB (Mathworks, Natick, Massachusetts) and DIAdem (National Instruments, Austin, Texas), the pressure data were plotted against time, and the peak pressure values were recorded.

Chapter 3: Results

3.1 Light Trap Data Analysis: Velocity

Since the light trap outputs its sensor data as voltage, the light trap data in volts for each spleen tests were graphed and the times for the black bar to pass were calculated. An example graph of the light trap data vs. time is shown below.



A B

Figure 6. The plots of light sensor voltage vs. time (a) The black dot indicates the time at the top of the bar. (b) The black dot indicates the time at the bottom of the bar.

X's from Figure 6 indicate time; therefore, the first X is equal to -0.02625 sec and the second X is equal to -0.0031 sec. The time took for the black bar to cross the light trap is around 0.02315 sec, and the velocity was calculated to be around 1.09 m/s. This process was repeated for every trial to calculate the impact velocity.

Below is the table of the experimental heights using the light trap and the calculated heights.

Table 4. Expected impact velocity, experimental height, and calculated heights

Expected Velocity (m/s)	Average Experimental Height (cm)	Calculated Height (cm)
1	8	5.096
2	23	20.39
3	47	45.87

3.2 Injury Analysis

After each impact tests, the peak pressures and impact energies were determined. The summary of injury results for all subjects is listed in Table 5.

Table 5. Summary of the results of the *ex vivo* spleen impact tests

Subject	Impact velocity (m/s)	Peak Pressure (psi)	Rate of Pressure change, $\dot{P}_{artery}$ (psi/s)	Impact Energy (J)	Damage
Spleen01	1.09	0.33 (A); 0.43 (V)	46.76	13.9	No damage
Spleen01	1.78	0.64 (A); 0.78 (V)	53.37	37.1	AIS 2
Spleen02	1.17	15.4 (A)	1057	16.0	AIS 3
Spleen03	1.32	17.9 (A); 17.4 (V)	1577	20.4	AIS 5
Spleen04	0.75	2.21 (V)	N/A	6.6	No damage
Spleen04	1.25	11.8 (V)	N/A	18.3	AIS 5

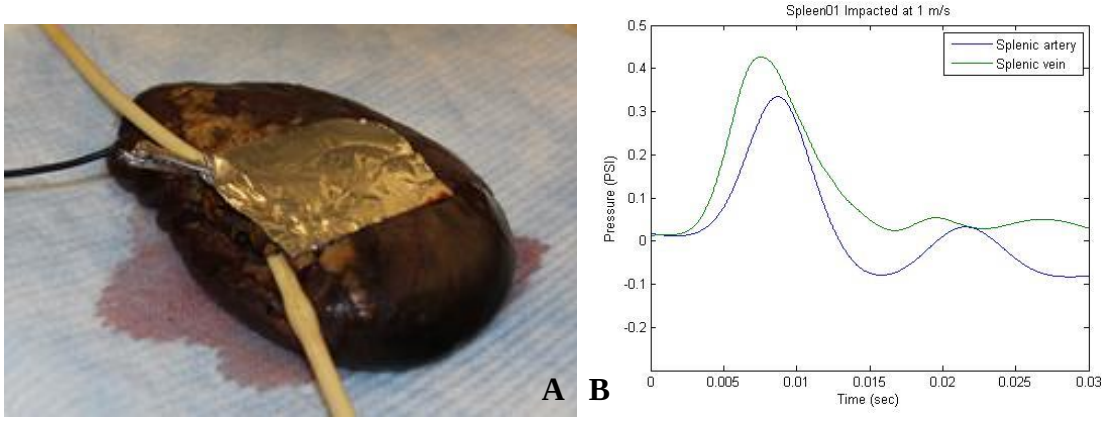


Figure 7. Spleen01 blunt impacted at a nominal velocity of 1 m/s (a) Spleen01 post-test image (b) Splenic artery and vein pressures vs. time

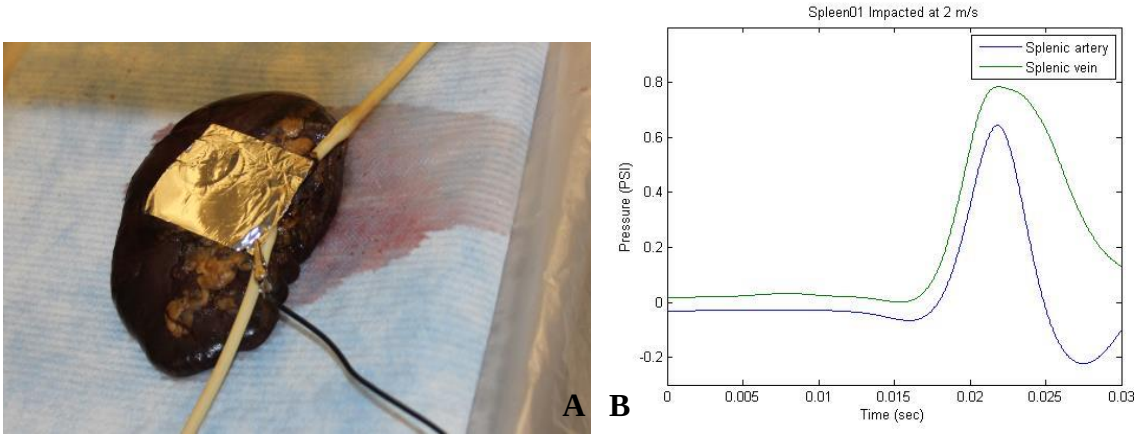


Figure 8. Spleen01 blunt impacted at a nominal velocity of 2 m/s (a) Spleen01 post-test image (b) Splenic artery and vein pressures vs. time

Spleen02 was only instrumented in the splenic artery with a foley catheter and pressure sensor due to the size of the vein. The splenic vein was tied off with a string to limit the fluid from exiting the vasculature and producing incorrect pressure data.

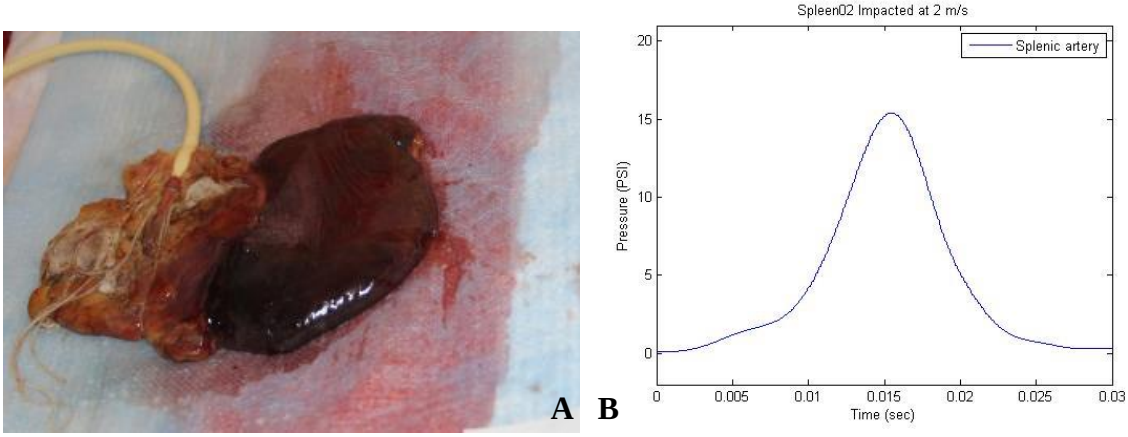


Figure 9. Spleen02 blunt impacted at a nominal velocity of 2 m/s (a) Spleen01 post-test image (b) Splenic artery vs. time

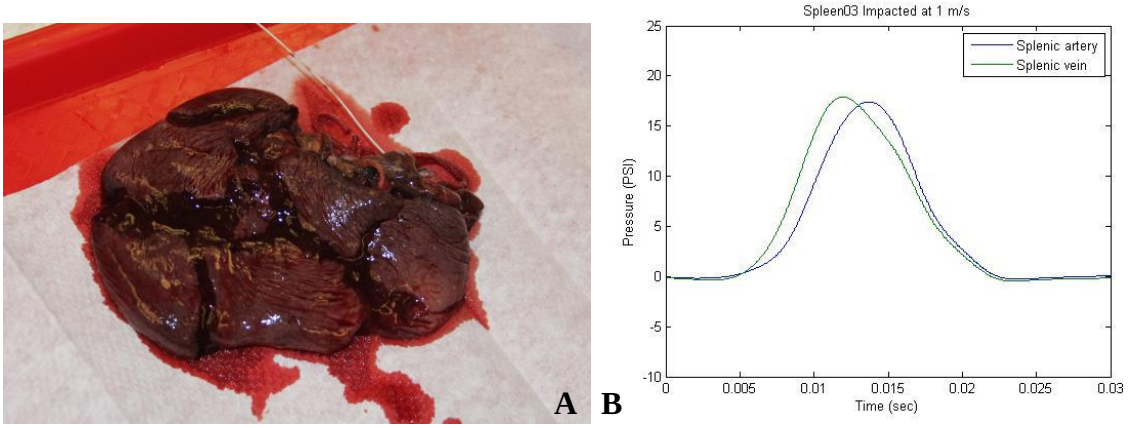


Figure 10. Spleen03 blunt impacted at a nominal velocity of 1 m/s (a) Spleen01 post-test image (b) Splenic artery and splenic vein vs. time

Spleen04 was only instrumented in the splenic vein with a foley catheter and pressure sensor due to the size of the vein. The splenic artery was tied off with a string to limit the fluid from exiting the vasculature and producing incorrect pressure data. Additionally, Spleen04 was impacted from a calculated velocity of 0.5 m/s in order to closely monitor the effects of velocity to injury.

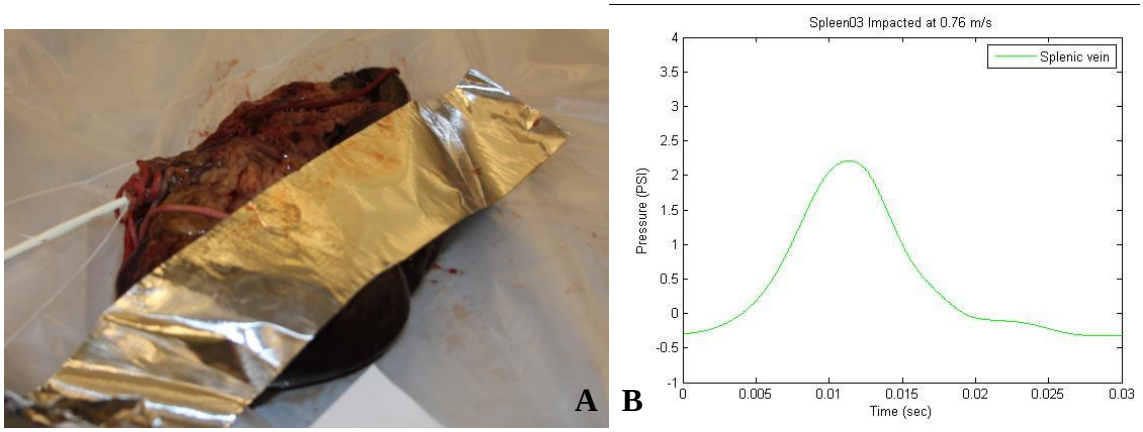


Figure 11. Spleen04 blunt impacted at a nominal velocity of 0.5 m/s (a) Spleen01 post-test image (b) Splenic vein vs. time

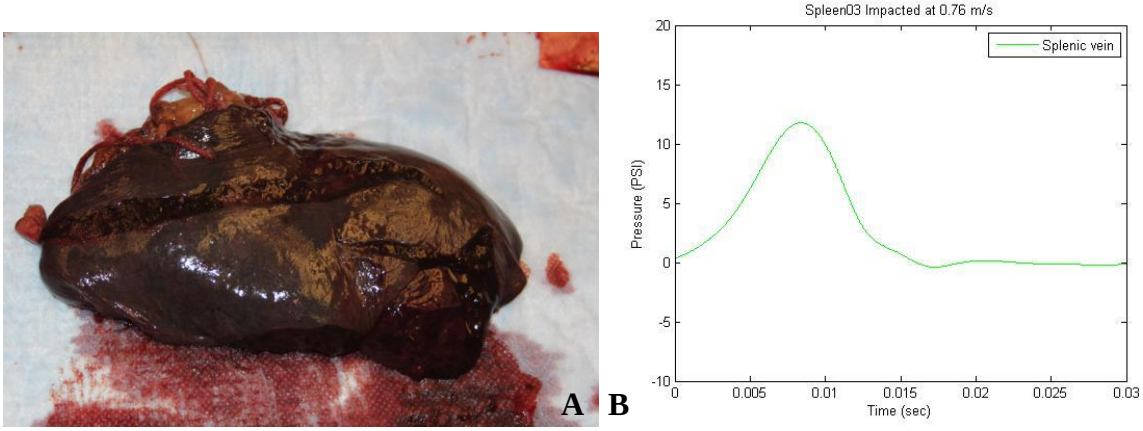


Figure 12. Spleen04 blunt impacted at a nominal velocity of 1 m/s (a) Spleen01 post-test image (b) Splenic vein vs. time

Specific injuries were measured, and their severities were determined. The injuries ranged from no visible damage to a critical damage severity, AIS 5. Table 6 contains specific injury descriptions for the *ex vivo* spleen impacts.

Table 6. Specific injury descriptions of the *ex vivo* spleen impacts

Subjects	AIS Number	Injury Descriptions
Spleen01	No damage	No visible damage on the surface was found
Spleen01	AIS 2	Laceration < 1 cm on the anterior face of the spleen

Spleen02	AIS 3	Laceration >3 cm on the posterior side of the spleen
Spleen03	AIS 5	Ruptured spleen
Spleen04	No damage	No visible damage on the surface was found
Spleen04	AIS 5	Ruptured spleen

Chapter 4: Discussion

4.2 Injury Outcomes

All *ex vivo* spleen subjects were tested until visible damages on the surfaces were seen (Table 6). Spleen01 and Spleen04 did not have any visible damages after impacts at velocities of 1.09 m/s and 0.75 m/s, respectively. The peak pressures were recorded as 0.43 psi and 0.75 psi, respectively, and $\dot{P}_{artery}$ was recorded as 46.76 psi/s for Spleen01. The impact energies peak pressures, and $\dot{P}_{artery}$ were the lowest for the above trials. On the contrary, Spleen03 and Spleen05 were ruptured, which can be defined as a critical injury, after impacts at velocities of 1.32 m/s and 1.25 m/s, respectively. The peak pressures were recorded as 17.9 psi and 11.8 psi, respectively and $\dot{P}_{artery}$ was recorded as 1577 psi/s for Spleen03.

During the second trial of Spleen01, the *ex vivo* spleen was impacted with the highest velocity of 1.78 m/s, but had a moderate injury of laceration less than 1 cm; however, the peak pressure for the above trial was correspondingly lower than the other trials with injuries greater than AIS 3. From such data, no conclusion regarding injury probability can be made from various impact velocities. However, according to the results, pressure and $\dot{P}_{artery}$ do correspond to the injury severities. Such data supports the assumption that the hydrostatic pressure is the major causation of injuries in motor vehicle crashes. the pressures were primarily used to determine injury probability since the focus of the current study is to relate the hydrostatic pressure of the spleens to injury severity in an *ex vivo* setting[9].

4.3 Injury Probability

In order to determine the probability of injury, injury risk greater than or equal to AIS 3 was determined for three variables, pressure, the rate of pressure change, $\dot{P}_{artery,}$ and velocity. For each trial, injury risk was given 0 if the injury severity was less than AIS 3 and 1 if the injury severity was greater than or equal to AIS 3. For pressure, injury risk was analyzed separately for splenic artery and vein. Injury risk greater than or equal to AIS 3 was plotted against vascular pressure (Figure 13).

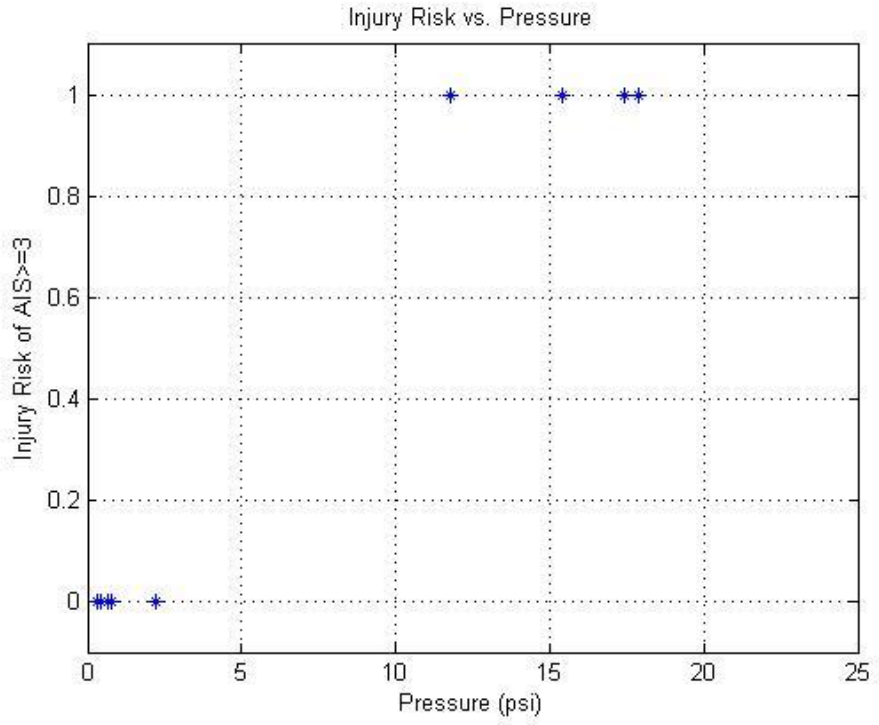


Figure 13. Injury of AIS>3 probability vs. vascular pressure

Injury risk was plotted against the rate of arterial pressure change, $\dot{P}_{artery}$ (Figure 14).

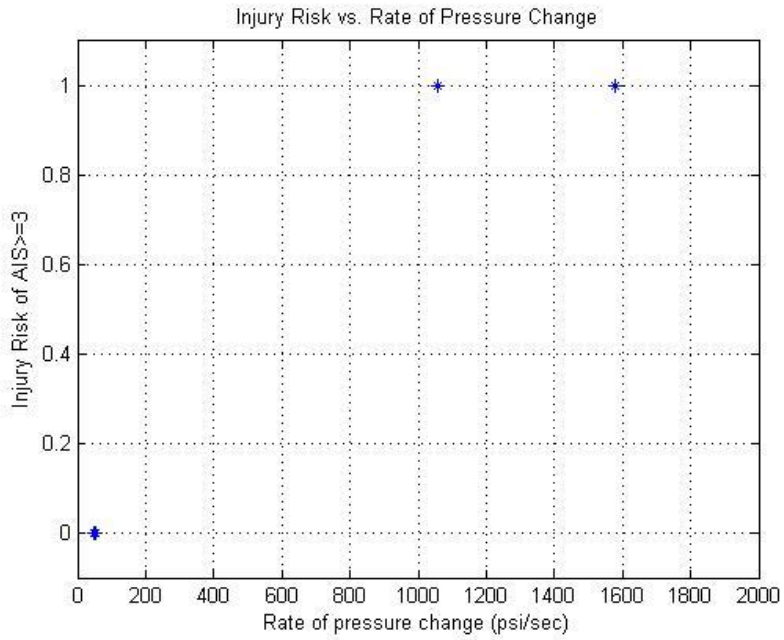


Figure 14. Injury of AIS>3 probability vs. rate of pressure change, $\dot{P}_{artery}$

Injury risk was plotted against the impact velocity (Figure 15).

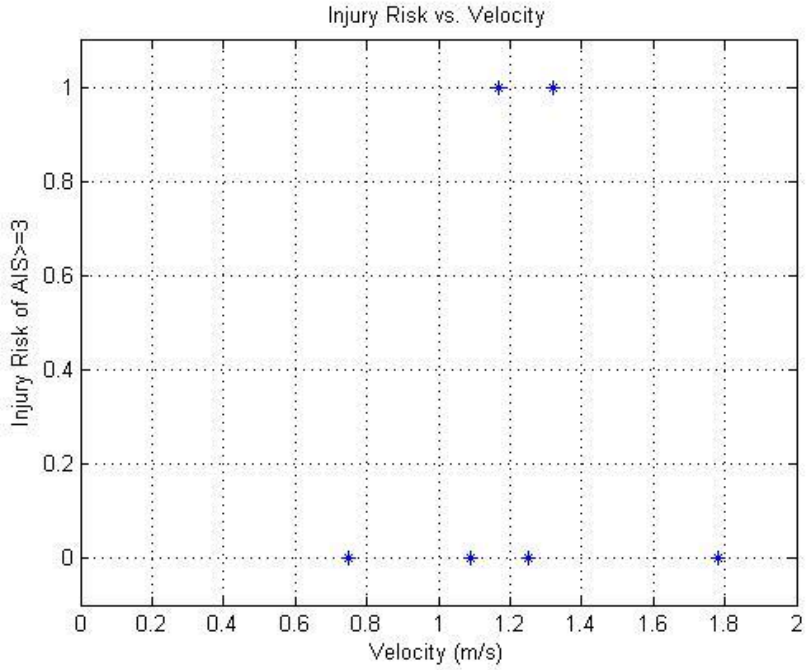


Figure 15. Injury of AIS>3 probability vs. impact velocity

Chapter 5: Limitations

The present study had limitations such as subject dependency, possible post-mortem tissue characteristic changes, limited data, *ex vivo* setting, and sample size. One of the major reasons for the irregular correspondence between impact velocity and injury could be due to subject variability. Since the *ex vivo* spleens were collected from 4 different individuals with different life styles and genetic expressions, the tissue stiffness and sizes may have been varied.

Another limitation is related to the number of days post mortem. The experiments were conducted generally four days after death, and the *ex vivo* spleens were frozen and thawed for about 24 hours prior to testing. The tissue characteristics may have been altered due to freezing and thawing.

The study also had limited data such as impact force and tissue pressure. The load cells used in these experiments produced inaccurate data to be useful for data analysis. Further testing could be performed with accurate load cells to determine the impact force and the *ex vivo* spleens can be further instrumented to measure the tissue pressure. More data on force and tissue pressure may help further define the injury risk.

The study was also limited to only *ex vivo* settings. From the study, it can be supported that hydrostatic pressure is one of the major causation of splenic injury. However, since the spleen is in a different environment *in vivo*, the current study's values cannot be compared with real-life motor vehicle collisions. For example, according to a study by Klinich, *et al.*, rib fractures were considered as one of the factors that produce

spleen injuries [4]. In this *ex vivo* study, no rib fractures were considered. A study using post mortem human subjects (PMHS) can be conducted to study the effects of blunt impacts in the *in vivo* environment that the spleens are in.

Lastly, the sample size of the study was limited four *ex vivo* spleens. Therefore, accurate injury risk curves against pressure, $\dot{P}_{artery}$, and velocity could not be produced. A further study with more samples can be conducted to produce more accurate injury risk curve for each variable.

Chapter 6: Conclusion

3 *ex vivo* spleens were tested and vascular pressures were measured from impact-induced hydrostatic pressure changes inside the spleen. The hydrostatic pressure changes in spleen showed correlation with injury, where severely injured spleens had higher peak pressure and $\dot{P}_{artery}$ (Table 5). Additionally, these initial experimental blunt spleen injuries produced in this testing compared well with those observed in motor vehicle crash victims [11,12].

Further testing can be conducted to verify these initial findings and produce more applicable results. Future works may include whole body post-mortem human surrogate (PMHS) testing with pressure transducers in the splenic vasculature if this trend continues. Since pressure is one of the major causation of splenic injuries, a preventative mechanism can be researched and developed to target decrease in pressure during motor vehicle crashes. Additionally, more analysis can be done to compare these testing energies with real-life crash energies.

Future works may also include producing more accurate test environments to assess injury through crash testing with dummies and simulating finite element analysis. The current crash dummies' abdominal regions do not include an accurate spleen model to relate pressure to injury; therefore, pressure sensors may be incorporated into the dummies to produce accurate injury risk during crash testing. Additionally, the properties of the spleen in finite element analysis models can be better defined to relate pressure and injury during simulations.

Appendix 1

Below is a MATLAB code to process the data for spleen01 as an example.

```
clc; clear all; close all;

%% Spleen 1

spleen01 = load('SpleenTest01.csv');
[B,A] = butter(2,0.07);

%% extract the millar data
time = spleen01(:,1);
timeplot = time(3000:13000);
millar = spleen01(:,2);
millarplot = millar(3000:13000);

% graph the millar data
% figure
% plot(time,millar)
% title('Pressure vs. Time')
% xlabel('Time (sec)')
% ylabel('Pressure (PSI)')
figure
plot(timeplot,millarplot) % shortened to show the pressure change
title('Focused Pressure vs. Time')
axis([0 0.02 -0.4 1])
xlabel('Time (sec)')
ylabel('Pressure (PSI)')
figure
plot(timeplot,filter(B,A,millarplot))
title('Filtered Focused Pressure vs. Time')
axis([0 0.02 -0.4 1])
xlabel('Time (sec)')
ylabel('Pressure (PSI)')

% Calculate pressure change
millarmin = min(filter(B,A,millarplot))
millarmax = max(filter(B,A,millarplot))
pressurechange = millarmax - millarmin

%% Calculate the rate of change in pressure (only in artery)

% from the graph, in the beginning and the end of the compression
includes
```

```
% the following data
time1 = 0.00615; %sec
pressure1 = 0.02597; %psi
time2 = 0.00961; %sec
pressure2 = 0.5171; %psi

% the rate of pressure change is
slope = (pressure2-pressure1)/(time2-time1)

%% extract the millar data -----millarsensor 02
time = spleen01(:,1);
timeplot = time(3000:13000);
millar2 = spleen01(:,3);
millar2plot = millar2(3000:13000);
[C,D] = butter(2,0.07);

% graph the millar data
figure
plot(timeplot,millar2plot) % shortened to show the pressure change
title('Focused Pressure vs. Time')
axis([0 0.04 -1 2])
xlabel('Time (sec)')
ylabel('Pressure (PSI)')
figure
plot(timeplot,filter(C,D,millar2plot))
title('Filtered Focused Pressure vs. Time')
axis([0 0.04 -1 2])
xlabel('Time (sec)')
ylabel('Pressure (PSI)')

% Calculate pressure change
millar2min = min(filter(C,D,millar2plot))
millar2max = max(filter(C,D,millar2plot))
pressurechange2 = millar2max - millar2min

figure
plot(timeplot,filter(B,A,millarplot),timeplot,filter(C,D,millar2plot))
legend('Splenic artery','Splenic vein')
title('Spleen01 Impacted at 1 m/s')
axis([0 0.025 -1 2])
xlabel('Time (sec)')
ylabel('Pressure (PSI)')

%% Extract the light trap data
time = spleen01(:,1);
timeplot = time(9200:10200);
Lighttrap = spleen01(:,6);
Lighttrapplot = Lighttrap(9200:10200);

% graph the millar data
figure
plot(timeplot,Lighttrapplot)
title('Light Trap vs. Time')
xlabel('Time (sec)')
ylabel('Light Trap Sensor (V)')
```

Below is a DIAdem code to process the pressure data for spleen01 as an example.

PSI indicates the artery pressure data, and PSI1 indicates the vein pressure data.

```
Option Explicit  'Forces the explicit declaration of all the variables in a script.
'-- Filter RAMXF to Class 60
Call CHNCFCFILTCALC("[1]/Time
axis","[1]/PSI","/FilteredSignal","CFC_60",0,"EndPoints",10) '...
XW,Y,E,CFCFILTTYPE,FIR100REMOVEBIAS,CFCPREEVENTTYPE,CFCFREEVALUE
Call ChnPropValSet("[1]/FilteredSignal","name","PSI CLASS60")
Call CHNCFCFILTCALC("[1]/Time
axis","[1]/PSI1","/FilteredSignal","CFC_60",0,"EndPoints",10) '...
XW,Y,E,CFCFILTTYPE,FIR100REMOVEBIAS,CFCPREEVENTTYPE,CFCFREEVALUE
Call ChnPropValSet("[1]/FilteredSignal","name","PSI1CLASS60")
```

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